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Research Article

Face-Referenced Measurement of Perioral Stiffness and Speech Kinematics in Parkinson's Disease

Shin Ying Chu,[a] Steven M. Barlow,[b] and Jaehoon Lee[c]

Purpose: Perioral biomechanics, labial kinematics, and associated electromyographic signals were sampled and characterized in individuals with Parkinson's disease (PD) as a function of medication state.
Method: Passive perioral stiffness was sampled using the OroSTIFF system in 10 individuals with PD in a medication ON and a medication OFF state and compared to 10 matched controls. Perioral stiffness, derived as the quotient of resultant force and interoral angle span, was modeled with regression techniques. Labial movement amplitudes and integrated electromyograms from select lip muscles were evaluated during syllable production using a 4-D computerized motion capture system.
Results: Multilevel regression modeling showed greater perioral stiffness in patients with PD, consistent with the clinical correlate of rigidity. In the medication-OFF state, individuals with PD manifested greater integrated electromyogram levels for the orbicularis oris inferior compared to controls, which increased further after consumption of levodopa.
Conclusions: This study illustrates the application of biomechanical, electrophysiological, and kinematic methods to better understand the pathophysiology of speech motor control in PD.

Parkinson's disease (PD) affects spinal motor systems involved in limb, gait, and respiratory functions (DeLong, 2000; Solomon & Hixon, 1993), and cranial motor systems involved in mastication, facial expression, vocalization, and speech motor control (Duffy, 2005). Hypokinetic dysarthria (HKD) is a motor speech disorder that affects 60%–90% of patients with PD and generally increases with disease duration (Logemann, Fisher, Boshes, & Blonsky, 1978). The hallmark characteristics of HKD are hypophonia, restricted pitch range, monoloudness, and variable production rate, generally attributed to a reduced range of articulatory movements (Darley, Aronson, & Brown, 1969), and a breathy and hoarse voice quality resulting in an overall reduction of speech intelligibility (Ackermann, Hertrich, Daum, Scharf, & Spieker, 1997; Adams & Dykstra, 2008; Darley, Aronson, & Brown, 1975; Fox & Ramig, 1997). Thus, HKD affects the clarity of speech by reducing mobility and scaling of the respiratory, phonatory, resonatory, and articulatory systems. Despite the prevalence of HKD and its impact, little is known about changes in underlying stiffness (rigidity) on perioral muscles and control. This study examined the predictive relation between perioral stiffness and select measures of motor output during syllable production, including electromyographic and kinematic patterning, in individuals with PD as a function of medication state.

There is a continuing debate in the PD literature on the origin of the motor control problems seen in this population. Specifically, the debate is whether the problem results from an underscaling of central motor commands (Berardelli, Rothwell, Thompson, & Hallett, 2001), the inability of the neuromuscular system to adapt quickly to the required force level (Weiss, Stelmach, & Hefter, 1997), or the inability of the muscular system to react with sufficient speed due to high postural stiffness (Ostry, Keller, & Parush, 1983). Studies of limb motor control in individuals with PD suggest an overall reduction in amplitude (hypokinesia) and velocity (bradykinesia) in addition to difficulty in planning and initiating movements (Morris, Iansek, Matyas,

[a]National Rehabilitation Center for Persons with Disabilities, Saitama, Japan
[b]Center for Brain, Biology and Behavior, Communication Neuroscience Laboratories, University of Nebraska, Lincoln
[c]Institute for Measurement, Methodology, Analysis and Policy, Texas Tech University, Lubbock
Correspondence to Shin Ying Chu: chu-shinying@rehab.go.jp

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& Summers, 1994; Viviani, Burkhard, Chiuvé, dell'Acqua, & Vindras, 2009). Thus, it is reasonable to hypothesize that basal ganglia dysfunction associated with PD may contribute to orofacial movement disorders in similar ways.

An abnormal increase in centrally mediated tonic drive on lower motor neurons translates to an increase in muscle stiffness, which is the clinical correlate of muscular rigidity in PD. Stiffness has been hypothesized to play a significant role in movement, including the regulation of end-point accuracy, force recruitment, and velocity scaling among articulatory systems for speech production (Gracco, 1994; Shaiman & Gracco, 2002). Measurements of jaw and lip stiffness reinforce the important role of biomechanics in speech and nonspeech movements (Chu, Barlow, Kieweg, & Lee, 2010; Barlow, Trotman, Chu, & Lee, 2012; Shiller, Laboissière, & Ostry, 2002). The pattern of jaw kinematic variation during simple consonant-vowel-consonant utterances has been associated with the spatial pattern of jaw stiffness (Shiller et al., 2002). Jaw perturbation during speech production has indicated that passive properties (stiffness) of the lips and jaw could contribute as a compensatory mechanism for achieving speech goals (Gomi, Honda, Ito, & Murano, 2002; Ito, Gomi, & Honda, 2000). The precise regulation of lip stiffness has also been found to be important for accurate production of fricative sounds such as [f] and [v] in healthy Japanese native speakers (Ito, Gomi, & Honda, 2004).

Parkinson's disease is frequently associated with elevated muscle activity at rest, which contributes to an increase in passive stiffness. Quantitative measures of passive stiffness in the perioral tissues have been sampled using a linear servomotor to impose sequential lateral tangential stretch of the oral angle in healthy adult women (Seibel & Barlow, 2007) and men (Chu, Barlow, & Lee, 2009). With this configuration, both women and men manifested a quadratic growth in interangle lip stiffness as a function of lip span. A case study in a patient with PD revealed a significant decrease in perioral stiffness after levodopa administration (Chu et al., 2010). Hunker, Abbs, and Barlow (1982) found that perioral stiffness among individuals with PD was positively correlated with electromyogram (EMG) in select muscles of the lower face and inversely related both to the magnitude of lip movement during speech (hypokinesia) and to spirantization of plosives due to errors in endpoint accuracy and incomplete articulatory gestures.

In order to determine the relation between muscle length (interangle span) and in vivo resultant force in the perioral region, active and passive forces were measured in the perioral system of healthy young adults (Barlow & Müller, 1991). Both the active and passive components of interangle force were found to be significantly related to interangle lip span as characterized by an exponential function over the range 25–70 mm, with adult men manifesting significantly higher maximum voluntary contraction levels compared to adult women.

Historically, pharmacological intervention using levodopa has been the most efficacious treatment for alleviating motor symptoms in PD (Bertoni, Prendes, & Sprenkle, 2001; Suchowersky, 2002). While the responsiveness of limb motor systems to levodopa has been widely studied, the corticobulbar speech system has received less attention. In general, the effect of dopaminergic stimulation on overall speech parameters and speech intelligibility in PD remains inconclusive (Pinto et al., 2004). Some authors have reported improvements across the speech parameters following levodopa administration, while others have found no effect of dopaminergic treatment on speech. Perceptual analyses of speech have documented improvements in articulation, pitch variation, voice quality (Wolfe, Garvin, Bacon, & Waldrop, 1975), tongue strength and endurance (De Letter, Santens, & Van Borsel, 2003), and speech intelligibility (De Letter, Santens, & Van Borsel, 2005) following levodopa administration. In an EMG study of labial muscles, Leanderson, Meyerson, and Persson (1971) reported that tonic hyperactivity of labial muscles decreased after medication, suggesting that levodopa normalized labial muscle activity. Conversely, levodopa therapy has also been documented to produce no improvement (Jiang et al., 1999; Solomon & Hixon, 1993) and a worsening of speech symptoms (Benke, Hohenstein, Poewe, & Butterworth, 2000; Goberman & Blomgren, 2003) in patients with PD.

The present study assessed the relation between passive resting-state perioral stiffness and the associated root-mean-square (RMS) of perioral EMG as a function of medication state (i.e., M-OFF vs. M-ON) among individuals with PD, as well as a secondary comparison to a control group of healthy age- and sex-matched participants (CG). In addition, we examined the relation between perioral stiffness and labial kinematics during syllable production at three speaking rates. Specifically, the following questions were addressed: (a) Are there differences in linear and quadratic components of the interangle stiffness as a function of medication state (M-OFF, M-ON) compared to CG? (b) Is passive-state perioral muscle activity dependent upon interangle span during the recoil phase to resting muscle length (distance between the corners of the mouth)? (c) Does this differ among M-OFF, M-ON, and CG? (d) Is perioral stiffness correlated to labial kinematics? For patients with PD, we hypothesized that interangle stiffness would be elevated in the M-OFF state compared to the M-ON state and CG. Perioral muscle activity was expected to remain relatively constant as interangle span increased. Finally, we hypothesized that a negative correlation would emerge between perioral stiffness and select measures of labial kinematics in the M-OFF and M-ON states.

Materials and Method

Participants

A total of 20 adults completed this study: 10 individuals with Parkinson's disease (four women, six men; age: M = 69 years, SD = 10.38) and 10 age- and sex-matched neurotypical adults (age: M = 70 years, SD = 8.93). Informed consent, approved by the University of Kansas Human Subjects Internal Review Board, was obtained from all

participants after the procedure had been fully explained. The physical characteristics of all participants are given in Table 1. The clinical profiles for the patients with PD are given in Table 2. CG and patients with PD did not differ in terms of age, gender, level of education, weight, height, head circumference, nasion-inion (measurement from the central point of the frontal suture to the external occipital protuberance), or lip resting span. Inclusion in the present study was limited to individuals with PD defined clinically by the presence of two out of three cardinal motor symptoms (i.e., tremor, rigidity, bradykinesia) and a positive response to levodopa as examined by a neurologist. Other inclusion criteria were no known history of neurological disease other than PD, no known history of any neuropsychiatric disorder, no known history of speech disorder, and normal/corrected visual acuity.

All participants completed the Speech Intelligibility Test (SIT; Yorkston, Beukelman, & Hakel, 1996), in which they read a series of 11 sentences. Audio recordings of these speech samples were scored by four first-year graduate students in speech-language pathology. No significant differences were found among participant groups for either speech intelligibility or speaking rate (see Table 3).

Protocol Overview

The study protocol included two test procedures. The first test (approximately 15 min) involved the measurement of perioral stiffness using the OroSTIFF device (Epic Medical Concepts & Innovation, Inc., Mission, KS). The second test (approximately 45 min) involved video motion capture of orofacial movements using a 4-D infrared digital-camera tracking system (Motion Analysis Corporation, Santa Rosa, CA). Both test procedures were completed in a single session for CG and in two sessions for the patients with PD, the first in the M-OFF state and the second in the M-ON state. All patients with PD were prescribed Sinemet (carbidopa-levodopa) by their neurologist at the time of this study. Because withholding medications for 12 h may cause physical discomfort for some patients with PD, the design of this study was not randomized. Each patient with PD arrived at the laboratory with her or his caregiver in the morning (8 a.m.) after withholding medication intake for 12 h prior to the testing. Once they finished the initial round of tests in the M-OFF state (by 9 a.m.), the patients with PD took their prescribed medications with a drink, and we repeated the stiffness and speech kinematics tests 1 h later when the participants were in the M-ON state. The procedure of testing the patients with PD in the M-OFF state followed by M-ON is consistent with the recommended protocol of the Core Assessment Program for Surgical Intervention Therapies in Parkinson's Disease for assessing the effect of intervention therapies in PD (Defer, Widner, Marié, Rémy, & Levivier, 1999). Thus, total session time for the patients with PD was approximately 3 h, compared to 1 h for CG participants.

Test 1: Perioral Stiffness

Passive nonparticipatory perioral stiffness was quantified in real time using a device developed in our laboratory known as OroSTIFF (see Figure 1). The OroSTIFF device is face referenced, which allows the participant freedom to move her head. This avoids the complications, discomfort, and potential artifacts associated with head restraint, especially in neurologic populations where dyskinesia can be an issue. The design, operation, and application of this biomechanical device have been detailed previously (Chu et al., 2010). The active cantilevers of the OroSTIFF device were coupled to the oral angles via contoured stainless-steel saddles. The body of the device was supported on the mental symphysis with a double-adhesive tape collar for vertical stabilization. This device incorporates a miniature pneumatic glass-cylinder actuator that is instrumented for pressure (Honeywell #26PCCFAG ± 15 psi) and an integrated displacement transducer (S-DVRT, MicroStrain®, Inc.) to encode the distance (aperture) between the corners of the mouth along the horizontal axis (Chu et al., 2010). The OroSTIFF device allows the tester to impose a slow interangle tissue stretch up to 20 mm relative to the resting lip aperture span. A fixed-leak resistance within the pneumatic actuator permits elastic recoil of the perioral tissues to return the instrumented cantilevers back to their resting lip span position. It is during this phase of passive recoil that reactive

Table 1. Physical characteristics of participants.

Variable	CG (*N* = 10) *M* (*SD*)	PD (*N* = 10) *M* (*SD*)	χ^2 or *t*	*p*	*w* or *d*
Age (years)	70.18 (8.93)	69.80 (10.38)	0.09	.93	.04
Gender			0.35	1.00	.00
Weight (lb)	176.10 (28.63)	175.88 (39.50)	0.01	.99	.01
Height (cm)	170.88 (10.89)	169.56 (12.08)	0.26	.80	.11
Head circumference (cm)	57.02 (2.03)	56.77 (1.56)	0.31	.76	.14
Nasion-inion (cm)	37.47 (1.72)	38.74 (1.92)	–1.56	.14	.70
Lip resting (cm)	50.70 (4.74)	52.37 (4.00)	–0.85	.40	.38

Note. In both CG and PD groups, six participants (60%) were men and four (40%) were women. *w* = Effect size for chi-square test. *d* = Effect size for independent-samples *t* test.

Table 2. Clinical features of individual patients with PD.

Participant	Sex	Age (years)	Years postdiagnosis	Hoehn & Yahr (1967) stage	UPDRS	Medication
PD 1	F	74	7	2.00	12	C-dopa/L-dopa
PD 2	F	71	7	3.00	39	C-dopa/L-dopa
PD 3	M	67	21	4.50	55	C-dopa/L-dopa entacapone
PD 4	F	75	6	1.00	19	C-dopa/L-dopa
PD 5	M	65	3	2.00	28	C-dopa/L-dopa pramipexole
PD 6	M	81	2	2.00	44	C-dopa/L-dopa
PD 7	M	61	3	2.00	45	C-dopa/L-dopa pramipexole
PD 8	M	72	5	2.50	69	C-dopa/L-dopa selegiline
PD 9	F	81	8	2.00	54	C-dopa/L-dopa
PD 10	M	46	2	2.00	38	C-dopa/L-dopa pramipexole
M (*SD*)			6.40 (5.58)	2.30 (0.92)	40.30 (17.2)	

Note. UPDRS = Unified Parkinson's Disease Rating Scale (Fahn, Ethon, & members of the UPDRS committee, 1987).

force and displacement signals were digitized (2000 samples/s, 16-bit resolution, ±5-V analog to digital converter [ADC]) and processed in real time to generate the stiffness function (defined as $k = N/mm$, where N is the recoil force between the oral angles and mm is the interangle distance).

Participants were seated in a comfortable chair and instructed to remain speechless during the perioral stiffness measurements. A 1-cm incisal bite block was molded (KERR Xtrude-XP) for each participant in order to stabilize the mandible. Interangle span was initialized to L_0 + 15 mm for all participants, where L_0 equals interangle lip resting span. A series of five interangle stretch trials was completed while simultaneously sampling force, displacement, and perioral EMG in real time with our custom software (OroSTIFF v.3.0.5) written in LabVIEW 8.0 (National Instruments Corporation, Austin, TX). Electromyograms were obtained using silver/silver chloride bipolar surface electrodes (4-mm diameter, 2-cm interelectrode distance, InVivo Metrics, Healdsburg, CA) placed on the left quadrant of the upper lip (orbicularis oris superior [OOS*m*]) and lower lip (orbicularis oris inferior [OOI*m*]). Biopotentials were conditioned with Grass P511 bioamplifiers (30 Hz–1 kHz bandpass, gain = 20 K, Grass Technologies, Warwick, RI), digitized at 2 kHz and digitally rectified and integrated (5-ms time constant). RMS integrated EMG (IEMG) levels for the two perioral-muscle recording sites were derived for CG participants and patients with PD (M-OFF and M-ON) during stiffness sampling.

Test 2: Lip Kinematics During Syllable Production

A four-dimensional optical motion capture system (Motion Analysis Corporation, Santa Rosa, CA) was used to track perioral movements during production of consonant-vowel syllables ("pa") as a function of production rate. Sixteen infrared reflective sphere markers (approximately 6 mm in diameter) were placed on the participant's lower face with double-sided adhesive tape (see Figure 2). In the present investigation, the peak amplitude associated with the inferior-superior movements of the upper lip (UL_y) and the lower lip plus jaw ($[LL+J]_y$) during alternating speech rates for the syllable "pa" was evaluated. Head motion was recorded with one reference array (consisting of four markers) centered at the forehead (see Figure 2). The motions of UL_y and $[LL+J]_y$ were calculated relative to the head coordinate system after the correction for head motion. This step ensures that extraneous head movements (e.g., dyskinesia) do not confound accurate registration of speech-related lip movements. All kinematic marker signals were sampled at 119.88 Hz and digitally low-pass filtered (f_{lp} = 10 Hz) using a zero-phase shift forward-reverse digital filter (Butterworth, eight-pole) written in a custom MATLAB program, Speech Movements and Spatial Histograms (Green, 2008).

A Sony electret condenser microphone (model ECM-DS30P) was attached to the participant's shirt collar to record the audio signal. The speech acoustic signal was digitized at a 4195.8-Hz sampling rate using a National Instruments

Table 3. Speech Intelligibility Test (SIT) means and standard deviations for PD and CG participants.

	PD (*N* = 10)				
SIT	M-ON	M-OFF	CG (*N* = 10)	*F*	*p*
Speech intelligibility (%)	96.1 (2.6)	96.4 (1.7)	97.3 (0.8)	$F(2, 27) = 1.15$	.33
Speech rate (words/minute)	176.4 (20.8)	177.7 (22.3)	183.3 (24.7)	$F(2, 27) = 0.26$	.77

Figure 1. OroSTIFF device used to assess perioral stiffness. S-DVRT = Subminiature-Differential Variable Reluctance Transformer.

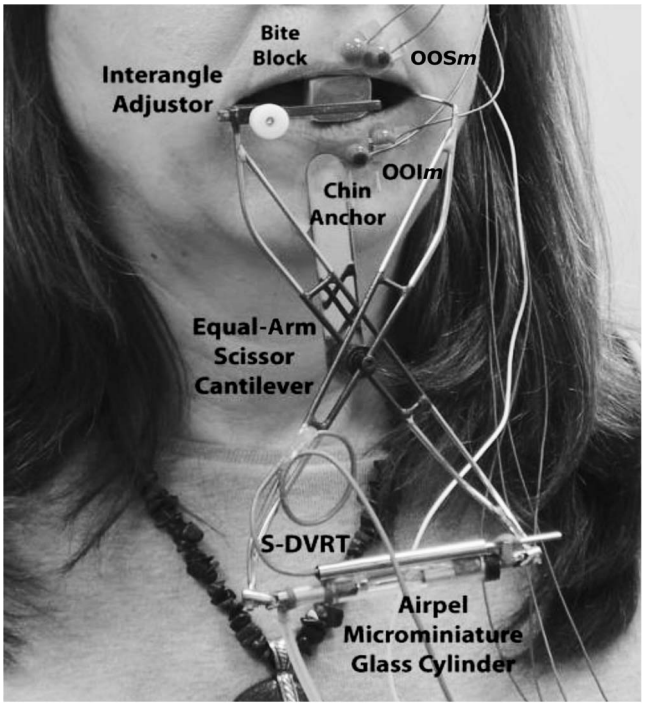
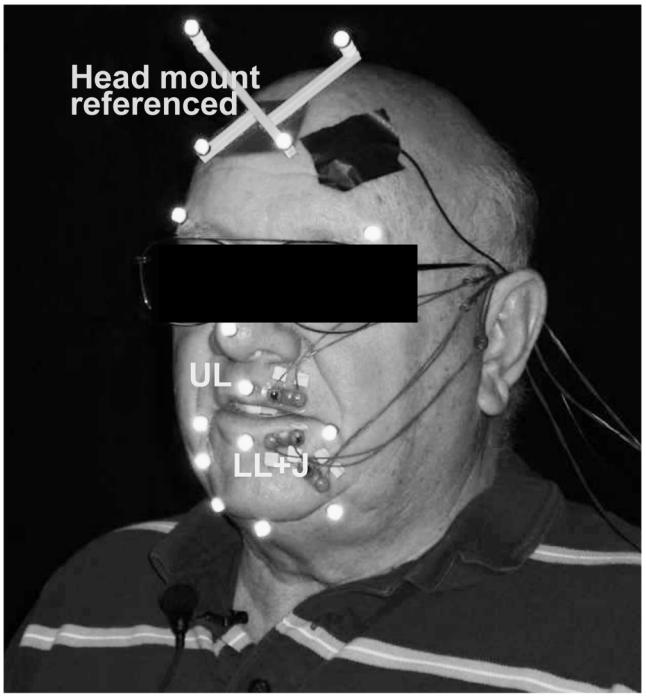


Figure 2. Marker placement for the motion capture system. UL = upper lip; LL + J = lower lip and jaw.



USB 6218 module integrated within the Motion Analysis data capture system. Participants were instructed to produce the "pa" syllable for 6 s at 2 syllables/s (six repetitions), 3.5 syllables/s (three repetitions), and 5 syllables/s (two repetitions) at their normal vocal intensity level. A metronome software program (Desktop Metronome, PA) was used to pace syllable production. The metronome signal was delivered to each participant via a headphone at an RMS vocal intensity level of 70 dB SPL.

Data Processing

Perioral Stiffness

Following an imposed pneumatic stretch of the mouth in the horizontal plane at the oral angles, stiffness coefficients (*N*/*mm*) were automatically calculated as the low-mass interangle yokes of the OroSTIFF device, powered by the force of tissue elastic recoil, returned to the participant's resting interangle span. As defined in our OroSTIFF (v.3.0.5) software, a stiffness coefficient was calculated as the change in force sampled at 1 mm intervals until the resting interangle span was attained. This process of imposing an interangle stretch and computing stiffness during passive recoil was repeated five times for each participant. Real-time display of stiffness coefficient versus span begins when three conditions are met simultaneously: span is greater than 0.5 mm, force is decreasing, and a positive slope exists for a 10-point linear fit of force versus span. Thus, the absolute number of stiffness measurements along the perioral recoil trajectory depends on the maximum interangle span achieved. The interested reader is referred to Chu et al. (2010) for a technical description of the bioengineering and mathematical computations involved in estimating interangle perioral stiffness using the face-referenced OroSTIFF device.

The Relation Between Perioral Stiffness and Labial Kinematics

A total of 30 consecutive syllable repetitions at three different rates were selected for analysis. To systematically select 30 productions of the "pa" syllable for the kinematic analysis, the following rules were established. The initial and final syllable within an utterance train were disregarded. The next five consecutive repetitions at 2-Hz productions, 10 repetitions for 3.5 Hz, and 15 repetitions for 5 Hz were included for analysis. To ensure that all starting and ending points of "pa" syllable trains were accurately selected, the $[LL+J]_y$ zero-crossing points on the velocity waveform (superior-inferior dimension, y-axis) were used to define the beginning (positive slope) and ending (negative slope) points of any given syllable train. Lip movement amplitudes were automatically extracted using a peak-detection routine (coded in LabVIEW 8.5) based on the quadratic fit algorithm to index velocity and displacement peaks. For the y-dimension, both closing and opening gestures were confirmed based upon the acoustic signal.

Statistical Analyses

Perioral Stiffness

To determine the pattern of perioral stiffness in M-OFF, M-ON, and CG conditions, a general linear mixed modeling was used to estimate random effects as well as fixed effects that occur at more than one level, accounting for dependency among observations within the participants. First, an unconditional means model (null model) was fitted to determine random variance structure. The fitted null model can be written as

$$\text{stiffness}_{ij} = \gamma_{00} + u_{oj} + e_{ij},$$

where $u_{oj} \sim N(0, \tau_{00})$ and $r_{ij} \sim N(0, \sigma^2)$ for trial i and participant j.

The stiffness score is expressed as the sum of an overall mean (γ_{00}), a random deviation from that mean (u_{oj}), and a random error (e_{ij}) associated with the ith trial in the jth participant.

Then the fixed effects for level-1 predictors (SPAN, SPAN^2), level-2 predictors (M-OFF vs. CG, M-ON vs. CG), and cross-level interactions were successively introduced into the null model with their random variance components (i.e., random effects). The inclusion of a fixed or random effect in the final model was determined independently by comparing model likelihood between two competing models (i.e., likelihood-ratio test [LRT]). For example, a random effect was kept if the LRT test result was significant, indicating that its inclusion significantly improves model fit. The fitted final model can be written as

$$\begin{aligned}\text{stiffness}_{ij} = {} & \gamma_{00} + \gamma_{10}\text{SPAN} + \gamma_{20}\text{SPAN}^2 + \gamma_{01}\text{OFF} \\ & + \gamma_{02}\text{ON} + \gamma_{11}(\text{SPAN} \times \text{OFF}) \\ & + \gamma_{12}(\text{SPAN} \times \text{ON}) + \gamma_{21}(\text{SPAN}^2 \times \text{OFF}) \\ & + \gamma_{22}(\text{SPAN}^2 \times \text{ON}) + u_{oj} + e_{ij}.\end{aligned}$$

The level-1 fixed effects (γ_{10}, γ_{20}) represent the linear and quadratic regression slopes of interangle span on perioral stiffness. The cross-level effects (γ_{11}, γ_{12}, γ_{21}, γ_{22}) represent the differences between M-OFF versus CG and M-ON versus CG in these linear and quadratic changes. Finally, in a supplemental analysis, the perioral stiffness changes were contrasted between two PD states (M-OFF vs. M-ON). The model effects were estimated using a restricted maximum likelihood method implemented in SAS 9.2 (SAS Institute, 2010).

Passive Stretch

The same modeling procedure described previously was used to confirm the nonparticipatory nature of muscle activity during interangle lip stretch.

Tonic IEMG RMS Level

A separate multilevel regression was conducted to compare the IEMG RMS level between M-OFF, M-ON, and CG conditions. The IEMG RMS means were estimated from the model with the level-2 predictors and the level-specific residual variance components. Then the estimated means were pairwise compared using a simulation adjustment for the multiple comparisons. This adjustment computed adjusted p values and confidence limits from the simulated distribution of the maximum or maximum absolute value of a multivariate t random vector (Edwards & Berry, 1987).

The Relation Between Perioral Stiffness and Labial Kinematics.

Pearson correlations were calculated to examine the relationship between perioral stiffness and movement amplitude, by gesture (opening and closing of "pa"), speech rate (2 syllables/s, 3.5 syllables/s, 5 syllables/s), articulator (upper lip, lower lip plus jaw), and condition (M-OFF, M-ON, CG). The stiffness scores at 12 mm of interangle span and the displacement distances were derived from each participant for the opening and closing gestures at the superior-inferior dimensions of the upper lip (UL_y) and the lower lip plus jaw ($[\text{LL+J}]_y$). The stiffness score at the 12-mm span were chosen because this was the maximum span from a patient with PD that we could derive.

Results

Perioral Stiffness

The parameter estimates from the fitted final model are shown in Table 4. The perioral stiffness function demonstrated a significant quadratic relation between imposed interangle stretch and resultant perioral force, $\widehat{\gamma_{20}} = 0.0003$, $t(2787) = 26.52$, $p < .001$. More importantly, the M-ON condition showed a significantly greater quadratic increase than the CG, $\widehat{\gamma_{22}} = 0.0001$, $t(2787) = 3.82$, $p < .001$. However, the quadratic increase did not differ between the

Table 4. Multilevel regression results for perioral stiffness.

Effect	Estimate	Standard error	*p*	*f*[a]
Intercept	0.0329	0.0053	.0000	
Span	–0.0044	0.0003	.0000	0.3794
Span^2	0.0003	0.0000	.0000	0.7482
Group				0.0179
M-OFF vs. CG	0.0006	0.0076	.9343	
M-ON vs. CG	0.0046	0.0076	.5559	
Span × Group				0.0632
M-OFF vs. CG	0.0014	0.0005	.0019	
M-ON vs. CG	–0.0002	0.0005	.6647	
Span^2 × Group				0.0731
M-OFF vs. CG	–0.0000	0.0000	.9508	
M-ON vs. CG	0.0001	0.0000	.0001	

[a]Effect size *f* was calculated based on type III test for the fixed effect.

M-OFF and CG conditions, $\widehat{\gamma_{21}} = -0.000001$, $t(2787) = -0.06$, $p = .95$. The supplemental analysis with only the PD states showed that the quadratic increase of perioral stiffness significantly differed between the M-ON and M-OFF conditions, with M-ON yielding a steeper stiffness function compared to M-OFF. This is consistent with the presence of high OOI*m* IEMG RMS in the M-ON state.

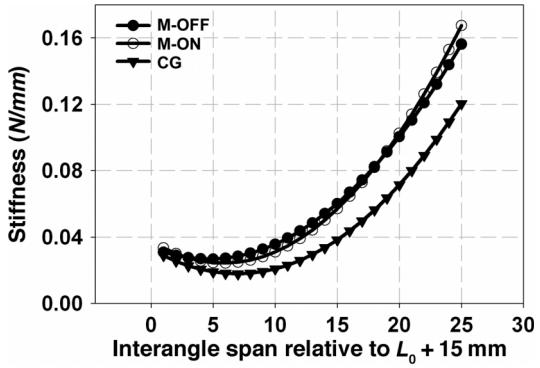
While there were 15 observations at 25 mm of interangle span in CG participants, only one observation was noted in the M-OFF condition at 25 mm of span, and none of the participants in the M-ON condition reached a 25-mm stretch. This is due to the increased lip stiffness levels among patients with PD, thus making it harder to impose interangle stretch with the OroSTIFF device. Mathematically, the patients with PD showed higher stiffness quadratic slopes than the CG participants, confirming an elevated stiffness in perioral tissues. Figure 3 illustrates the estimated perioral stiffness changes derived from the final multilevel regression model. The final model is

$$\begin{aligned}\widehat{\text{stiffness}}_{ij} = {} & 0.0329 - 0.0044 \times \text{SPAN} \\ & + 0.0003 \times \text{SPAN}^2 + 0.0006 \\ & \times \text{OFF} + 0.0046 \times \text{ON} + 0.0014 \\ & \times (\text{SPAN} \times \text{OFF}) - 0.0002 \\ & \times (\text{SPAN} \times \text{ON}) - 0.000001 \\ & \times \left(\text{SPAN}^2 \times \text{OFF}\right) + 0.0001 \\ & \times \left(\text{SPAN}^2 \times \text{ON}\right). \end{aligned} \quad (1)$$

Passive Stretch

The estimated distribution of the pooled IEMG RMS values at the OOS*m* and OOI*m* muscle recording sites is shown in Figure 4. The parameter estimates from the fitted final model are shown in Tables 5 (OOS*m*) and 6 (OOI*m*). No significant (linear) slope was found for the OOI*m* IEMG, $\widehat{\gamma_{10}} = 0.0316$, $t(2792) = 0.69$, $p = .49$, indicating that the tonic drive to the perioral muscles remained constant during interangle stretch. For the OOS*m* IEMG, the M-ON condition showed a significantly greater linear increase than CG, $\widehat{\gamma_{12}} = 0.0248$, $t(2790) = 2.43$, $p = .05$, while the linear increase did not differ between M-OFF and CG, $\widehat{\gamma_{11}} = 0.0149$, $t(2790) = 1.43$, $p = .15$. Although all the conditions showed a slight increase in the OOS*m* IEMG during perioral stretch, this increase was less than 2 μV over the 25-mm increase in interangle span. Therefore, there was no significant evidence of reflexive and/or voluntary activity during the imposed perioral stretch, thereby confirming the nonparticipatory nature of the experimental task.

Figure 3. Estimated perioral stiffness means for M-OFF (closed circle), M-ON (open circle), and CG (triangle).



Tonic IEMG RMS Level

A significant condition effect was found for M-ON versus CG, $t(8) = 2.84$, $p < 0.05$, on OOI*m* IEMG RMS level. The estimated means were significantly different between M-ON and M-OFF (adjusted $p < .05$) for both OOS*m* and OOI*m* IEMG RMS levels. In addition, M-ON showed a greater level of OOI*m* IEMG RMS ($M = 24.72 \pm 33.91$ μV) compared to OOS*m* IEMG RMS ($M = 6.31 \pm 3.41$ μV), paired $t(878) = -15.98$, $p < .001$. Overall, the PD conditions showed a greater OOI*m* IEMG RMS level compared to CG, and the elevation of IEMG RMS became greater following administration of levodopa.

The Relation Between Perioral Stiffness and Labial Kinematics

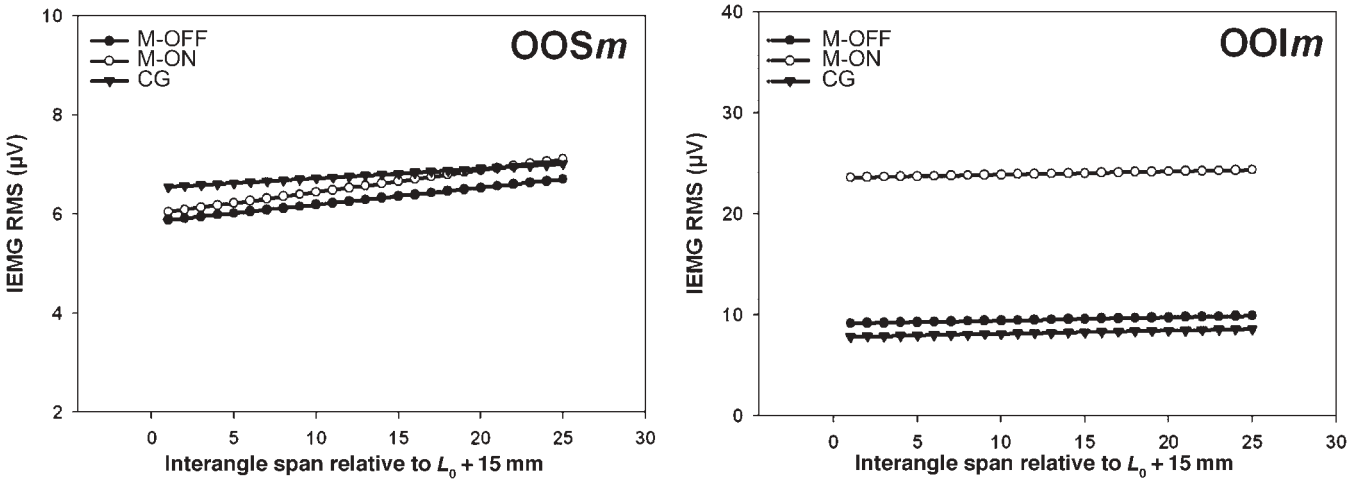
For CG and M-OFF, no significant correlations were found between perioral stiffness, gestures (opening, closing), and speech rates (2 syllables/s, 3.5 syllables/s, 5 syllables/s). However, there was a significant correlation between perioral stiffness and both UL_y opening amplitude, $r(8) = .64$, $p < .05$, and UL_y closing amplitude, $r(8) = .69$, $p < .05$, at the speech rate of 5 syllables/s in M-ON (see Table 7). These findings confirm a relation between perioral stiffness and UL_y movement displacement, but only at the highest speech rate used in the present study.

Discussion

Muscular Stiffness in Perioral Muscles

Multilevel regression modeling of perioral stiffness revealed significant differences in perioral stiffness across the M-OFF, M-ON, and CG conditions, indicating that clinical rigidity associated with PD affects orofacial muscles. The present findings on perioral stiffness are consistent with those of previous studies on orofacial stiffness in patients with PD (Caligiuri, 1987; Seibel & Barlow, 2007). Perioral stiffness in the upper and lower lips has been found to be greater in four patients with PD than in CG participants (Hunker et al., 1982). Seibel (2003) reported that patients with PD showed significant differences of lateral tangential interangle lip stiffness when measured with a digitally controlled linear servomotor. Specifically, four out of seven patients with PD exhibited improvements in their perioral stiffness functions during the M-ON state. It is apparent that stiffness (elevated rigidity) in PD is not limited to limb or axial muscles, nor a manifestation limited to muscles

Figure 4. The distribution of the mean integrated electromyogram (IEMG) root-mean-square (RMS) values for upper lip (OOS*m*) and lower lip (OOI*m*) during nonparticipatory conditions for M-OFF (closed circle), M-ON (open circle), and CG (triangle).



endowed with muscle spindle afferents, since perioral muscles do not contain these mechanoreceptors (Folkins & Larson, 1978; Lovell, Sutton, & Lindeman, 1977). The present findings dispel the notion derived from studies of limb function that increased stiffness is principally due to increased gamma motor drive to muscle spindles (Burke, Andrews, & Lance, 1972; Rushworth, 1964).

The fact that perioral muscles are attached directly to the integument of the skin in the lower face, along with the absence of spindle end organs (Folkins & Larson, 1978), points to the probable role of cutaneous and deep mechanoreceptor activity in modulating proprioceptive cues in the regulation of movement in the perioral region (Pinto et al., 2004). The slow-adapting Aβ mechanoreceptor described as a pseudo-Ruffini corpuscle ending has been shown to encode stretch and directional information with a best frequency from DC to 10 Hz, which makes this nerve ending well suited to encode the dynamics of perioral movements (Johansson & Olsson, 1976). A high density of stretch-sensitive slow-adapting mechanoreceptive units that exhibit similar physiological properties as the Ruffini ending has been found in the transitional zone of the lip region (Johansson, Trulsson, Olsson, & Westberg, 1988; Nordin & Hagbarth, 1989) and may serve a proprioceptive role in the perioral region (Barlow, 1987, 1998). The present study did not include specific tests of kinesthesia or proprioceptive capacity, and thus any conclusion regarding the theory that the bradykinesia and rigidity that manifest in patients with PD are related to abnormal processing of the mechanoreceptor sensory inputs is speculative (Tatton, Eastman, Bedingham, Verrier, & Bruce, 1984).

Regardless of medication state, the elevated perioral stiffness demonstrated by the patients with PD was consistent with the findings by Caligiuri (1987) and Hunker et al. (1982). While these two studies quantified labial stiffness with a linear function, our investigation shows that the stiffness coefficient was dependent on stretch displacement and modeled by a quadratic function. Moreover, Caligiuri (1987) and Hunker et al. (1982) measured the perioral stiffness resulting from imposed (inferior-superior) displacements of the lips at midline, whereas our study imposed stretch in the horizontal plane, between the corners of the

Table 5. Multilevel regression results for OOS*m* IEMG RMS.

Effect	Estimate	Standard error	*p*	*f*[a]
Intercept	6.5268	1.0984	.0000	
Span	0.0194	0.0064	.0024	0.1368
Group				0.0000
M-OFF vs. CG	−0.6806	1.5539	.6730	
M-ON vs. CG	−0.5268	1.5538	.7433	
Span × Group				0.0381
M-OFF vs. CG	0.0149	0.0104	.1520	
M-ON vs. CG	0.0248	0.0102	.0156	

Note. OOS*m* IEMG RMS = Root-mean-square of integrated electromyogram for the upper lip.

[a]Effect size *f* was calculated from the results of type III test for the fixed effect.

Table 6. Multilevel regression results for OOI*m* IEMG RMS.

Effect	Estimate	Standard error	*p*	*f*[a]
Intercept	7.8251	3.9248	.0607	
Span	0.0316	0.0460	.4925	0.0000
Group				0.3928
M-OFF vs. CG	1.3112	5.5066	.8178	
M-ON vs. CG	15.7337	5.5062	.0212	

Note. OOI*m* IEMG RMS = Root-mean-square of integrated electromyogram for the lower lip.

[a]Effect size *f* was calculated from the results of type III test for the fixed effect.

Table 7. Correlation between perioral stiffness and movement amplitude.

Amplitude variable	Articulator	CG *r*	M-ON *r*	M-OFF *r*
2-Hz opening	UL_y	–.13	.18	–.20
2-Hz opening	$[LL+J]_y$	.25	–.25	–.44
2-Hz closing	UL_y	–.11	.32	–.45
2-Hz closing	$[LL+J]_y$	.26	–.27	–.43
3.5-Hz opening	UL_y	–.04	.20	–.06
3.5-Hz opening	$[LL+J]_y$	.30	–.21	–.19
3.5-Hz closing	UL_y	–.06	.26	–.07
3.5-Hz closing	$[LL+J]_y$	.29	–.22	–.19
5-Hz opening	UL_y	–.33	.64*	.26
5-Hz opening	$[LL+J]_y$	.11	–.01	–.14
5-Hz closing	UL_y	–.32	.69*	.22
5-Hz closing	$[LL+J]_y$	.12	–.02	–.15

*$p < .05$.

mouth, thereby increasing interangle span. This displacement trajectory was more akin to the transverse fiber orientation of the orbicularis oris muscle fiber complex (Blair & Smith, 1986; Müller & MacLeod, 1982; Müller, Milenkovic, & MacLeod, 1985). Considering the underlying dynamics (position end point, force end point, rate of force change, velocity, etc.) of the orofacial muscles, we modeled the perioral stiffness with second-order equations to more accurately represent the low-mass viscoelastic perioral system. When modeling the perioral stiffness function, we found that the M-OFF function versus the CG function was significantly different using a linear fit, whereas M-ON versus CG was significantly different when applying quadratic fits. The findings also suggest that a quadratic increase in perioral stiffness with interangle span was the prevalent pattern across all conditions. This quadratic slope is likely to change indicative of increasing perioral stiffness with disease progression. We have noticed this previously in a case study of a patient with moderate–severe PD in the M-OFF state who manifested a perioral interangle stiffness slope approximately 7 times greater than that of a CG participant (Chu et al., 2010). Thus, biomechanical modeling using nonlinear regression techniques is well suited to capturing subtle changes in stiffness regulation in the complex perioral system (Müller et al., 1985).

One may presume that dopaminergic treatment would reduce perioral stiffness in patients with PD, yet those in this investigation demonstrated a greater quadratic function slope in perioral stiffness after the administration of levodopa. These findings are in apparent contradiction to previous studies of orofacial biomechanics and electromyograms that focused on the neurophysiology of speech articulators and found a beneficial effect of levodopa (Leanderson et al., 1971; Nakano, Zubick, & Tyler, 1973). However, the finding in the current study that perioral stiffness increased during the M-ON state corresponds with the increased central drive as reflected in the OOI*m* IEMG. Just as control of limb stiffness functions as an important strategy for maintaining adjustments during movements, changes in perioral stiffness and tonic IEMG may be used to maintain positional control and stability of the mandible during speech production and mastication.

Verification of the Passive Stretch

Because PD is a central progressive neuromotor disease, the design of the current study was to test nonparticipatory "passive" stiffness during a series of low-velocity imposed perioral stretches. Electrophysiological monitoring of perioral IEMG was used to verify that voluntary and/or reflexive muscle activation did not contaminate the stiffness measure. Although the OOS*m* IEMG RMS activity levels showed an increased activation pattern across interangle span for all groups (see Figure 4), these increases were less than 2 μV. This confirmed the absence of reflex activity during perioral stretches and suggests that the growth in stiffness as a function of interangle span is presumably due to a combination of elastic forces generated by muscle and connective tissue and an elevated central tonic drive to motoneurons within the facial motor nucleus.

Greater Tonic IEMG RMS Activity in PD

Although it was demonstrated that patients with PD showed excessive IEMG activity at rest, consistent with the classical notion, it was also observed that IEMG in those participants was greater than in CG participants. This difference increased nearly threefold after the administration of prescribed levodopa treatment, especially in the OOI*m*. It is also possible that elevated background tonic IEMG in early stages of PD contributes to and modulates postural alignment so that it does not also resist intended movements. Similar to the muscular stiffness that is essential to the regulation of posture and interjoint coordination (Nichols, 2002), skilled motor behavior, such as speech, also requires dynamic modulation of muscle stiffness in order to achieve greater prediction of movement and endpoint accuracy. Measurements of jaw stiffness during speech and nonspeech tasks have shown the ability of typical participants to modify jaw stiffness in order to maintain postural stability in the presence of external loads (Shiller, Houle, & Ostry, 2005). For example, up-regulation of jaw stiffness has been shown to decrease kinematic variability during speech (Shiller et al., 2002). Our finding that up-regulation of perioral stiffness is associated with slight improvement in labial kinematics performance in the M-ON condition (compared to M-OFF) represents a powerful means by which the nervous system may attempt to maintain mechanical stability during speech production. Comparing the upper and lower lip IEMGs for PD and CG conditions, the OOI*m* consistently showed greater activity than the OOS*m*. This observation is consistent with the hypothesized antigravity function of the OOI*m* for postural control of the lower third of the face (Barlow & Rath, 1985; Seibel & Barlow, 2007) compared to its upper lip counterpart. Regulation of stiffness within the perioral system, a springlike property of the neuromuscular system, is thought to play an important role in sharpening the system's

behavior for rapid alternating movements associated with speech production (Ito et al., 2004). Hence, the basal ganglia, in addition to regulating muscle tone and energizing muscle activation in the limb system, are likely to modulate postural control among orofacial muscles.

Relation of Stiffness and Hypokinesia in the Upper Lip

A significant negative correlation was found between magnitude of perioral stiffness and range of labial movements, indicating a trading relation between the orofacial postural control system and voluntary perioral movements during speech production in PD. The present findings expand on our understanding of the relation between perioral stiffness and hypokinesia in individuals with PD, specifically in the M-ON condition at the speech rate of 5 syllables/s. The finding that perioral stiffness and hypokinesia did not show a significant correlation at the $[LL+J]_y$ and at the slow rate of speech production raises the following concerns. First, it suggests that a fairly large change in speaking rate (i.e., 5 syllables/s) may be required before a correlation in perioral stiffness will be observed. Second, it suggests that there may be differences across structures (i.e., UL_y and $[LL+J]_y$) in terms of the extent to which a given rate of speech will be associated with a particular change in stiffness profile. The OroSTIFF device we utilized to sample the interangle perioral stiffness measured a composite of force and interangle displacement during perioral stretches. Therefore, no data are available to determine the passive stiffness–hypokinesia correlation relative to upper lip and lower lip independently. Future study will consider an enhanced design to address this question.

Limitations

The current findings are at odds with a previous report on labial stiffness in PD (Caligiuri, 1987). Using a linear motor transducer, Caligiuri reported increased labial stiffness for upper and lower lip muscles in 12 patients with PD; however, stiffness was sampled in a midline lip compression vector rather than by imposing a tangential stretch of the orbicularis oris muscle fibers directly. Caligiuri found no apparent relation between labial stiffness and the decrement in the range of lip movement. The inconsistencies may be attributed to the radically different methods of stiffness sampling and analytic methods employed by these two studies. Stiffness coefficients were derived in the previous study, while the present study utilized a definite perioral stiffness score at 12 mm of interangle displacement, as well as nonlinear regression modeling over the full range of stretch imposed on the orbicularis oris muscle-tissue complex. Future study could model the relationship of perioral stiffness and hypokinesia using high-level statistical methods, such as structural equation modeling, in a larger cohort of more severely affected patients with PD.

Conclusions

This investigation showed promise in developing a quantitative metric of perioral stiffness for revealing changes in tonic motor neuron drive that may be dependent on medication status and disease progression. The ability to evaluate the efficacy of the medication state using the face-referenced OroSTIFF device supports the view of Müller et al. (1985) that assessment of perioral stiffness could provide a useful set of biomarkers to clarify the effects of progressive neuromotor disease and to test hypotheses concerning articulatory dynamics. With a relatively high stiffness–hypokinesia correlation ($r = .69$) shown in our study, the inclusion of a larger sample of participants at more advanced stages of PD is likely to shed light on the perioral stiffness–hypokinesia relation. This investigation showed that a quantitative metric of stiffness is useful in revealing changes in tonic motor neuron drive that is likely to change due to medication status and disease progression.

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